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SOME PRINCIPLES OF HUMAN GENETICS

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THE field of human genetics has now increased to vast dimensions, having fundamental bearings on both medicine and anthropology. Comparison with animal genetics shows many differences, both in methods and results. The absence of experimentation in man makes it necessary to rely upon pedigrees as the raw materials on which much of our knowledge of human heredity rests, although the sib-pair method makes it possible to obtain some genetic results from observations of sibs in a single generation and twin-studies also yield important evidence. It has sometimes been stated that human genetics is essentially population genetics. But while it is important to know the frequency and distribution of all recognized genes in any human population, yet it appears that the pedigree must remain the fundamental unit of study. This is because an abnormality with essentially the same phenotypic expression may be due to a dominant gene in one family, a recessive gene in another pedigree and a sex-linked gene in a third. The statistical pooling of these different groups inevitably leads to misleading results.

Possible explanations of the state of affairs in which different types of inheritance are involved for essentially the same condition, have been discussed elsewhere (Gates, 1946a, b), but the widespread occurrence of this triad of methods of inheritance of many human abnormalities,

such as megalocornea, microphthalmia, peroneal atrophy or even albinism, makes it impossible to lump together all the pedigrees of each condition in a population and derive from it a statistically significant result. It is not enough to inquire, "How is diabetes or cataract inherited?" but "How are they inherited in each particular family pedigree." The assumption is still widespread that albinism is universally recessive, yet there are perfectly clear pedigrees in which it is confined to males and inherited as a sex-linked recessive. It is well-known that there are more human albino males than females, and many attempts have been made to explain this excess of affected males, except the obvious explanation provided by the fact that in the sex-linked pedigrees only males are affected. A single dominant pedigree of albinism is known. In a family in which, we will say, only two brothers are known to be albinos, it would be impossible to say without further evidence whether the gene involved is simple recessive or sex-linked. There is similarly evidence, not yet conclusive, that schizophrenia is generally recessive but occasionally dominant in inheritance, while in *diabetes mellitus* both dominant and recessive forms are recognized.

While it is thus true that the same medical condition frequently shows different methods of inheritance, thus complicating the statistical study of the corresponding gene distributions in the population and necessitating the grouping of the pedigrees together according to the method of inheritance in each, yet a triad of inheritance methods is by no means universal. Although there are exceptions to any rule which may be formulated, one may say that anatomical abnormalities are usually inherited as dominants, while metabolic deficiencies such as alkaptanuria, ochronosis, steatorrhoea, porphyria, von Gierke's glycogen disease or amaurotic idiocy are recessive. In these metabolic defects, as in the X-ray metabolic mutations of Beadle and Tatum in Neurospora, and similar biochemical mutations produced recently in *Bacillus sub-*

tilis, Penicillium species and *Ophiostoma,* a particular enzyme is absent which is normally concerned in bringing about a specific reaction in the organism. This enzyme is believed to be either the gene itself or an immediate product of the gene, set free in the cytoplasm of the cell. Similarly, in human metabolic defects there is probably only a short series of chemical changes between the particular gene and the specific enzyme which results from its presence.

As regards the usual dominance of anatomical abnormalities which are not lethal in effect, polydactyly has often been regarded as a universal dominant. Snyder (1929) formerly concluded from an extensive pedigree of a Negro family, that although dominant in whites polydactyly was probably recessive in Negroes. In a large collection of Negro pedigrees not yet published are included 27 pedigrees of ulnar polydactyly (an extra little finger). In 22 of these pedigrees the condition is dominant, with a total of 213 cases. The sibships, where one parent was affected, give a total of 180 affected: 175 normal children where a 1:1 ratio is expected; but the sex-ratio of the affected is peculiar, the total of affected (202) being 123 ♂ : 79 ♀. The large excess of affected males indicates greater penetrance in the male than in the female sex. In all these families there is only an occasional skip of a generation (*i.e.,* lack of penetrance). These are all Negro pedigrees except one Hindu family from British Guiana with 37 cases in six generations. The remaining five pedigrees include two single sporadic cases of polydactyly. These might be instances of recessive polydactyly or possibly of fresh mutation. The other three pedigrees include 11 cases and probably represent recessive inheritance, though they might be families with low penetrance of a dominant gene.

Thus the conclusion is clear from these 27 pedigrees, that polydactyly is usually dominant in Negroes as it is in Caucasians or Hindus, but that in a few families the gene is either recessive or a dominant of low penetrance. The

indications are also that the recessive form is no more severe than the dominant, and that the latter is no more extreme in the homozygous than the heterozygous condition.[1] The high frequency of polydactyly in Negroes is well recognized—an interesting example of a marked racial difference in the frequency of a particular gene.

As pointed out in detail elsewhere (Gates, 1946b), it is usual for the recessive form of any abnormality to be most extreme, the dominant form mildest and the sex-linked form intermediate in expression. The most likely explanation of this appears to be on the basis of a difference in the relative *strength* of the genes involved. In the dominant condition a single gene representing the defect is pitted against the corresponding normal gene, the result being a relatively mild form of the disease, with later onset. In the recessive form the gene is present twice, without any countervailing normal gene. The result is a much more severe form of the disease, generally with earlier onset of symptoms. However, as the effect of a single recessive gene is almost or completely suppressed by its normal allele, it is evident that the recessive gene must have less strength than the corresponding dominant.

As regards the sex-linked gene, it is present in the X-chromosome of the male and there is no corresponding normal gene in the Y to mitigate its effect, so the sex-linked condition is usually more severe than the dominant form but less so than the recessive. These relationships are particularly clear in the case of peroneal atrophy. In the dominant form the mean age of onset is about 19 and the patient does not become crippled. In the sex-linked form the muscular atrophy begins in boys at about 15 and is more severe, an occasional woman transmitter being also partially affected. The recessive form is so severe that children three to five years of age may have difficulty in walking, the mean age of onset is about 11 years, and patients are severely crippled by the time they reach twenty years of age. The dominant form

of this disease does not interfere with reproduction, but in the severe recessive form reproduction must be rare although the condition is not lethal. Nevertheless there is no indication that one form is being transformed into the other by any kind of selection, or that any change in dominance is taking place. In Bell's (1935) monograph on peroneal atrophy she found 55 dominant pedigrees, six sex-linked and 32 more or less clearly recessive. Thus the dominant gene was much the most common, the sex-linked occurring in only six pedigrees out of 96.

Since the recessive form of peroneal atrophy must largely prevent reproduction, it must be replenished from time to time by fresh mutations. Now, what can we say regarding the germinal relationship of these three forms? An obvious possibility regarding the sex-linked form would be that its gene has been translocated from an autosome. If this were the case there might be two degrees of the sex-linked form, as there is an equal possibility that the stronger dominant or the weaker recessive gene should be so translocated. Since the symptoms of the sex-linked form are more severe than those of the dominant, it would appear that the latter may have been the one translocated from an autosome; but of course there is no evidence that such a translocation has taken place, and it is quite possible that the sex-linked gene has arisen as a mutation *in situ* in the X-chromosome.

Much has been made of the theory of modifiers in seeking to explain changes of dominance, and this hypothesis appears to be theoretically adequate to explain many cases in animal genetics where a gradual change from recessive to dominant is believed to have occurred. For example, R. A. Fisher's (1935) theory of the origin of dominant conditions in the domestication of fowls. Although I know of no evidence for such a change in man through the accumulation of modifiers, we can probably not exclude the possibility at the present time. Of course, some cases of "skipping a generation" might be due to the presence in the individual of such a dominance modi-

fier. However, cases of skipping are usually not sporadic, but they occur with a frequency which appears to be characteristic of the pedigree and could be better accounted for either by a weakness in the gene or by a condition widely present in the genotype of the family in question.

The human pedigrees we can collect seldom go back ten generations, usually only three or four. The effect we are considering could hardly be expected to be perceptible in so few generations involving so few individuals. Anticipation (earlier onset) appears to be frequently only a statistical effect in many cases, resulting from the way in which the data are collected, although there are certain cases which may not be amenable to this explanation, but we do not propose to discuss that subject here.

Some recent experiments of Dobzhansky and Spassky (1947) with laboratory cultures of *Drosophila pseudoobscura* extend over 50 generations of flies and yield results which may have a bearing on this question of the relation between the corresponding dominant and recessive disease-genes in man. These experiments, which occupied over four years, are far too extensive to summarize here, but they have an important bearing on questions of how populations may change through selection under different conditions. Seven strains were made up homozygous for certain second or fourth chromosomes and kept in mass culture for fifty generations under conditions of crowding or over-population. Five of these homozygous strains showed improvements. In the other two there was no change or slight deterioration in viability. In one strain the homozygotes had, at the beginning of the experiment, deformed legs and wings and elongated abdomens. At the end of fifty generations the deformed legs and wings had been replaced by normal, but the long abdomen—an innocuous character—had been retained.

[1] This conclusion is confirmed by a recent pedigree of unilateral (left hand) hexadactyly (Barrer, 1948) with 19 cases in five generations of a Belgian family. Two affected parents had six children, all affected in the same way, although one of them at least should have been homozygous for the gene.

Seven other strains were made up in exactly the same way, but in each generation the males were treated to heavy doses of X-rays. After fifty generations three of these lines showed striking improvement in viability, two showed some improvement, one line was unchanged and one showed deterioration. In four of these lines the rate of development of the homozygotes was accelerated, but in some it was slowed down. Others showed reduced fertility or structurally abnormal homozygotes. Chance thus plays a role but there is a tendency towards improvement in both the treated and untreated homozygous cultures under the action of selection.

On the other hand, in strains in which these particular chromosomes were kept in a balanced condition and the individuals not overcrowded, the recessive mutations arising, most of which are deleterious, are sheltered from natural selection and thus accumulate in the heterozygous condition, whereas in the homozygous strains deleterious mutants are eliminated while beneficial ones are multiplied in the population. Among the balanced strains which were not treated with X-rays, three developed lethals in the balanced chromosomes, one showed a slight improvement and three were unchanged. In general, most of the homozygous lines improved while most of the balanced lines degenerated.

An early experiment of Marshall and Muller (1917) has perhaps a more immediate bearing on our problem of dominance and recessiveness. Homozygous strains of the recessive mutant balloon, in *Drosophila melanogaster,* were bred for fifty generations. Parallel cultures were kept heterozygous for fifty generations. The end result was that the mutant characters were more extreme in the cultures which had been kept heterozygous than in those which were homozygous for the balloon mutation. It may be supposed that modifiers lessening the effect of the gene would tend to be selected under competition in the homozygous strains, while modifiers intensifying these genic

effects could have been selected in the heterozygous strains.

It is not clear, however, why selection should act in this way in the heterozygotes; and it is equally difficult to see why in fifty generations of a human pedigree selection should act in a consistent way on modifiers of a recessive gene, ultimately changing it to a dominant. It seems necessary to return to the view that in human pedigrees the dominant and recessive genes for a particular mutant condition differ at least in strength, and there is evidence in certain cases that they also differ in other respects. It would simplify matters if they could be shown to be alleles; but the evidence, so far as it goes, is definitely contrary to such a conclusion. The ultimate reason for these dominant and recessive pairs remains obscure. They are not mimic genes in the ordinary sense, because they differ in strength and therefore in their method of inheritance.

It is well-known that in some dominant mutations, such as bar eye in Drosophila, there is a duplication in several crossbands or genes of a salivary chromosome. Double duplication of this zone enhances the bar eye effect, and with loss of the duplication the eye reverts to the recessive normal. Here is a possible basis for some of the cases in which dominant and recessive forms of the same abnormality are found in different pedigrees. The fact that the cubitus interruptus mutation changes from recessive to dominant in the presence of translocations between chromosome-IV and the Y in Drosophila indicates another possible basis for this dominant-recessive pair in man. Dry (1944) finds in New Zealand Romney sheep that in different strains the so-called N-type of coat is dominant, recessive or multifactorial in different stocks. He suggests by analogy that a gene duplication has taken place in the dominant form. When the newer tissue-culture methods are applied to man a cytological basis for some of these differences in dominance may perhaps be found.

Another feature of human genetics which needs only

to be mentioned here is the fact that a number of genes for abnormal conditions are almost or quite confined to one human race. For example, Cooley's anaemia (thalassemia) is found in the races around the eastern Mediterranean, sicklemia in about seven per cent of American Negroes and also in Africa in a higher percentage, amaurotic idiocy in Jews, Marchiafava's disease in Italians, favism in Sicilians and Southern Italians. The gene for haemophilia seems to occur most frequently in the Germanic peoples and, like the blood groups, various other genes determining hereditary diseases differ in frequency from one race to another. The rarity of *erythroblastosis foetalis* in Chinese results from the fact that nearly 100 per cent are positive for the Rh agglutinogen. On the other hand, it has recently been found that about 36 per cent of the Basques in Argentina are Rh negative, while it is well known that this peculiar race has a lower percentage of the B blood group than any other people in Western Europe.

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INHERITANCE IN CROSSES OF JERSEY AND HOLSTEIN-FRIESIAN WITH ABERDEEN-ANGUS CATTLE. II. COLOR AND WHITE SPOTTING[1]

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INTRODUCTION

IN the cattle breeding experiments, carried out by the Department of Genetics, University of Wisconsin, where polled self-colored Aberdeen-Angus animals were crossed to horned Jerseys and to horned and spotted Holstein-Friesians, color and markings of the foundation and crossbred animals were carefully recorded. With a few exceptions each individual was described and photographed at certain intervals, and hair samples were taken of the calves for a study of the kind and distribution of pigment. In a previous publication (I: Figs. 1–5) on the inheritance of horns and shape of skull mating charts were presented for the experimental herd. On these charts also the color and the extension of white markings were denoted for each animal. The reader is referred to these charts, and to the description of foundation animals, notes on the recording of data, etc. In the present paper the data on color and white spotting will be presented and discussed, particularly in relation to the hypotheses on color inheritance in cattle, published by Ibsen (1933) and by Bogart and Ibsen (1937).

Bogart and Ibsen found two different pigments in the hair of cattle, *viz.* the clumped black and the diffuse, nongranular red pigment. All breeds of cattle are assumed to be homozygous for the red (*RR*) as well as for the

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black pigment (*BB*). Black hair, such as in Angus and Holstein cattle, contains closely packed black clumps and is due to the extension gene (*E*). Partial extension of black pigment, the "blackish" color found in Jerseys and Ayrshires, is supposed to be due to a gene *Bs,* acting as a modifier on *B*. Ibsen (1933) postulates modifiers of *Bs, M* causing much black and *L* little black, *M* being dominant in the male and *L* in the female. Another gene, *Br,* is supposed to cause brindling in joint action with *Bs,* i.e. black or blackish stripes on an otherwise red animal. The dilution of pigment in such breeds as Jersey and Guernsey would be due to a recessive gene (*ii*), or in some cases perhaps to a dominant gene (*D*). The Jerseys are supposed to be homozygous for a recessive gene (*ww*) for "whitening," causing the whitish band surrounding the usually black muzzle, and also some white or whitish hairs on the belly and udder and on the inside of the ears and rear legs ("fawn"). Ayrshires and Shorthorns carry a dominant gene (*Pl*) for "pigmented legs;" the pigmentation is concentrated around the hoofs and extends upward, in contrast to the Holstein and Guernsey breeds where pigmentation spreads downward past the knee and hock. Such spotted breeds as Holstein, Ayrshire, Guernsey and, in some cases, Jersey are homozygous for recessive white spotting (*ss*); an incompletely dominant gene (*Lw*) tends to reduce the amount of white whereas animals of the genotype *lwlw* may be almost entirely white. Ibsen also postulates a number of dominant genes for white spotting; of those genes only *In,* causing inguinal white, will be mentioned here.

Where fairly good evidence is produced for definite genes for color or white spotting, we will use the same symbols as Ibsen (1933) and Bogart and Ibsen (1937). We are hesitant, however, to accept some of their postulates due to lack of evidence.

The Angus × Jersey cross.

Three of the foundation Angus animals used in this

cross (1*A* ♂, 3*A* ♀, and 4*A* ♀) were solid black with the exception of a small white spot, or a few white hairs, in the inguinal region. The cow 16*A* had no white in the inguinal region but she had a few white hairs in the switch. One cow, 3*A*, is recorded as having a reddish tinge on the left thigh and on both flanks, but a rusty appearance is not at all uncommon in black breeds, depending on age, exposure and other environmental conditions.

The Jersey bull 2*A* and the cow 5*A* were gray fawn, the bull being somewhat darker than the cow. The cow 6*A* was a cinnamon fawn and the bull 13*A* was of rather dark color, with white spots on flank and in the elbow region. All except 13*A* had a light ("whitish") band around the muzzle, which is characteristic of most Jerseys (*cf.* Fig. 1).

The F_1 generation.

The F_1 generation consists of 12 animals, all black, and none showing the light band around the muzzle. All F_1 animals but three (7*C*, 9*B* and 19*A*) showed a small amount of white in the inguinal region, as did most of the purebred Aberdeen Angus parents. The cross Angus ♂ × Jersey ♀ and its reciprocal gave the same result, no consistent difference in shade of color being observed in the two groups of F_1 animals and there was likewise no consistent color difference between the sexes. Most of the F_1 calves showed a brownish or dark fawn color on the underline, inside of legs and in neighboring regions, but as the animals grew older the majority (7 out of 12) became solid black. Two steers, 8*C* and 9*B*, castrated at approximately two months of age, showed at time of slaughter (15 and 22 months old respectively) some degree of fawning. This was rather pronounced on the steer 9*B*, not only on the belly and legs but also on the body. He also displayed an indistinct brindling. The bull 19*A*, slaughtered at 18 months of age, had a rusty color and showed dilution on the belly. Two cows, 8*B* and 14*A*, also showed a similar dilution to some ex-

tent. Tongue color is recorded for only seven animals, and in all these cases at least the anterior part of the tongue was black. The muzzle was black in all cases.

The results show that the black color of the Aberdeen-Angus breed is dominant to the Jersey color, although the expression of dominance may not always be complete, probably due to modifying factors. Our results agree with earlier observations on F_1 animals, obtained in crosses between the same breeds (Parlour, 1913; Kuhlman, 1915; Gowen, 1918; Hooper, 1921; and Olson, 1928).

The F_2 generation.

The classification of the F_2 animals into distinct color groups is very difficult. Two (11*D* ♂ and 12*A* ♀) are described as being without fawning; 15*A* as black but having a reddish patch near the left shoulder, and 21*A* as black with a tendency to reddishness. The steers 11*C*, 12*C* and 21*B* and the cows 11*A* and 12*E* were black but showed some rustiness in the coat and more or less distinct color dilution (fawn) on inside of thighs and on underline. They were all lighter in color as calves and darkened gradually as they approached maturity. None of these animals had the light muzzle band. The heifer calves 12*D* and 15*D* both died at about three months of age. They were described as being Jersey colored with light muzzle band, but would probably have darkened with age. The steers 11*F* and 15*E* were described as dark fawn with light band around muzzle; 15*D* and 15*E* were distinctly brindled. Although the color variation in F_2 is rather continuous, ranging from solid black Angus color to dark Jersey fawn, 9 out of the 13 live-born animals may be classified as black and 4 as being of Jersey color. If this classification is correct the results approximate the 3:1 ratio as nearly as would be expected with such small numbers. Of 4 aborted or stillborn calves, 3 (11*B* ♀, 11*E* ♂ and 12*B* ♂) may be classified as black. The fourth, 15*B* ♂, is described as "uniform dark." If this also is to be considered a black, the total F_2 ratio becomes 13 black to 4 Jersey color.

Six of the live-born F_2 animals were self-colored, and seven had some white spotting in the inguinal and umbilical regions. The amount of white spotting segregating out in F_2 was not in excess of that found in the foundation animals. All had black muzzle and the tongue was at least partially black in all cases where recorded.

Backcrosses to Jersey bulls.

Four calves (Fig. 3) were raised from two F_1 cows, 7*A* and 8*B*, backcrossed to the Jersey bull 29*A*. This bull was of uniform dark fawn color with small white markings on flank and tail. Two of the calves from 8*B* (42*A* ♀ and 42*C* ♂) were black with brownish fawn color on belly and inner thigh, characteristic of some of the F_1 and most of the F_2 animals. The third (42*B* ♂) was very dark fawn with light muzzle band. The steer 42*C* had considerably more white than is usually found in the Jersey breed. Both hind feet were white almost to the hock, the left front foot had a small spot of white, and almost the whole underline was white. The cow 42*A* had a big splash of white in front of the udder. Steer 44*A* (dam: 7*A*) was a diluted red of almost Guernsey color; umbilical region and inside of legs were light cream-colored and horns and hoofs were yellow. This animal had a light muzzle band. The tongue was flesh-colored in 3 of the backcross animals and dark pigmented in one.

Four F_2 cows (11*A*, 12*A*, 12*E* and 21*A*) were backcrossed to Jersey bulls. The rusty black F_2 cow, 11*A*, produced with a dark fawn Jersey bull (13*A*) a brindle bull calf (22*A*), of Jersey-like color and with light muzzle band. With the grayish fawn Jersey bull 24*A*, this cow gave a bull calf, 27*A*, of diluted red color with light muzzle, horns and hoofs. The solid black F_2 cow 12*A* produced, by three different Jersey bulls, 4 black calves, two of which showed the dilution characteristic of the crossbred animals. The cow 30*B* had white hind feet and no black pigment on the tongue. Cow 12*E*, a full sister of 12*A*, produced by the Jersey bull 29*A* a brownish black heifer; and the black F_2 cow 21*A* produced by

the same bull 3 black calves showing some dilution. Presumably 12*A* and 21*A* were homozygous for black.

The cow, 42*A*, herself a backcross from an F_1, and 30*B*, backcross from an F_2, were both bred back to their Jersey sire, 29*A*, and produced respectively calves 65*A* ♂ and 66*A* ♀. These were both of Jersey color with light muzzle band but they had no black pigment on the tongue; 66*A* had some white on the underline but for 65*A* there are no records of white markings.

Discussion

The fundamental genetic difference between the color of the Angus and Jersey breeds is that the former breed is assumed to be homozygous for *E* and the latter for its allele (*e*), and in addition most Jerseys are homozygous for partial extension of black (*Bs*). The *Bs* gene produces black pigment at a lower rate than *E*, and the red pigment is therefore completely covered by black only in certain areas particularly on the head and legs. The different shades of Jersey colors are probably due to a number of modifiers influencing the concentration of black pigment, and also to factors diluting the red pigment. This would explain the continuous variation of color shades in F_2, tending to obscure the simple genetic relation between *E* and *Bs*. The Jerseys used in our experiment did not carry genes which actually dilute black pigment. The lighter appearance in some of the F_1 and F_2 animals is due to the different degrees of fawning. In the cross between black Angus and red and white Holstein, or red Angus × black and white Holstein, all F_1 animals raised to maturity became intense black—the black pigment thus completely covering the red. For a thorough study of the inheritance of Jersey color it would be necessary to raise a fairly large number of F_2 animals, or backcrosses, to maturity because the colors of full grown animals and calves are not comparable.

According to Ibsen (1933), the Jersey breed is homo-

zygous for the recessive "whitening" gene (*w*). The degree of whitening is very variable. In our F_1 generation the light band around the muzzle is entirely suppressed, and in F_2 it is present only in animals which show the Jersey fawn color. In the Brown Swiss breed the light muzzle band and faded color inside of ears and legs is much more consistent and less variable than in the Jerseys. This may be associated with the general darker color of the Brown Swiss, for in Jerseys the muzzle band and fading in the inguinal region are most noticeable in the darker animals.

Brindling occurs in some of the segregates from the Jersey × Angus cross. In our F_2 generation two out of four Jersey-colored animals were distinctly brindled, and in backcrosses of F_2 cows to Jersey bulls one of two fawn segregates was brindled. The dam of the brindled steer 15*E* was black and could therefore not have shown the brindling factor if carrying it. According to Kuhlman (1915) two brindled animals among four Jersey-colored were obtained in the F_2 generation in the English Angus × Jersey cross referred to before. Cole (1925) suggested that brindling is due to the joint action of two genes, one for partial extension of black pigment (*Bs*) and one for the concentration of this pigment in stripes or brindles (*Br*). In black animals (*EE* and *Ee*) the effect of *Br* and *Bs* is masked by the action of *E*. Our data are too limited to furnish any evidence of the relationship between *Bs* and *Br* as to allelism, dominance or interaction. The brindling factor was presumably introduced to the Jersey × Angus cross by some of the foundation Angus animals.

In many of the F_2 and in some of the F_1 calves from the Jersey × Angus cross there is a distinct darkening with increasing age until maturity is reached. This is noticed especially in the Jersey-colored, dark fawn, animals, but is also apparent in some animals which at maturity become practically self black. Some F_1 and F_2 calves are, however, born as black as Angus calves of

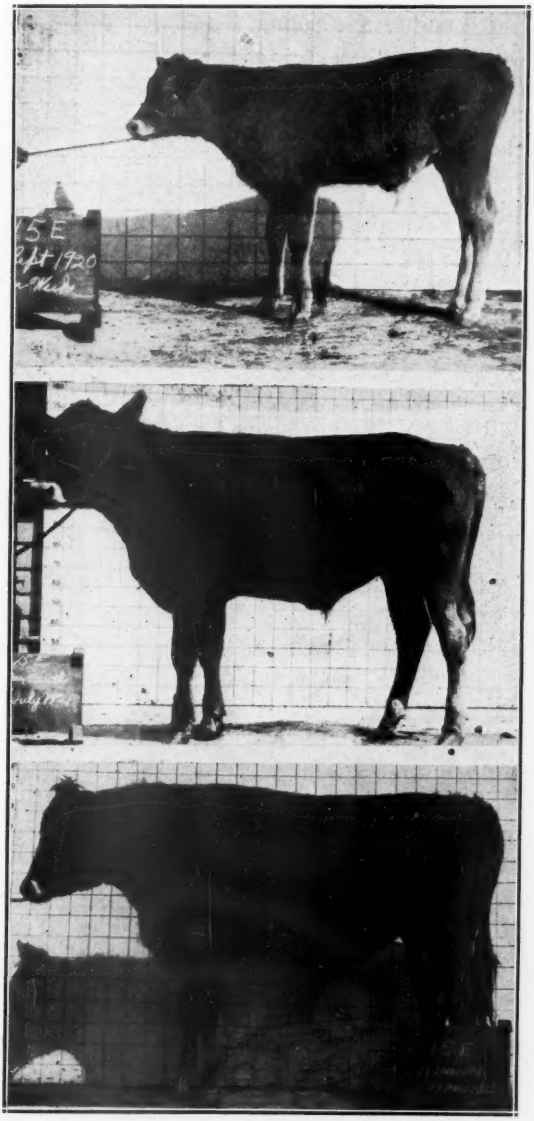

FIG. 1. Age darkening of the Jersey × Angus F_2 steer 15*E*. Age: 2, 14 and 21 months respectively. Color: brindle brown with the light band around the muzzle, which is a characteristic of the Jersey breed.

the standard color. No sexual dimorphism or effects of castration were noted in the darkening of crossbred calves. Figure 1 shows the age darkening in the F_2 steer 15*E*. This animal was distinctly fawn at birth but became dark brindled at maturity. That the black Angus color is not always completely dominant in F_1 is probably due to the great variation in shades of Jersey color, which variation must be assumed to have a genetic basis. The black pigment seems to creep in gradually during the growth of the animal, thus more and more completely covering the red.

The Angus × Holstein cross.

All the Aberdeen Angus bulls (50*A*, 54*A*, 80*A* and 113*A*) and two of the cows (16*A* and 18*A*) used in the production of the F_1 generation were black, whereas the other cows of this breed (32*A*, 35*A*, 36*A* and 37*A*) were red segregates from registered black parents. These segregates were of clean red color without any intermingling of black; 35*A* and 36*A* were of a rich, deep shade; 32*A* was considerably lighter. All except 32*A* had a small amount of white in the inguinal region. The black Angus cow 18*A* also had some inguinal white, but her dam 16*A* had only a few white hairs in the switch. No description is available of the amount of white in the Angus bulls, but if any was present it was confined to the inguinal region.

The foundation Holstein bulls (20*A*, 33*A*, 39*A* and 67*A*) were black and white, (Fig. 2) but all the Holstein cows (34*A*, 40*A*, 41*A*, 45*A*, 52*A*, 60*A* and 98*A*) were red and white segregates from registered black and white parents. An examination of the photographs of the red and white cows reveals a type of marking that is certainly not characteristic of Holsteins, but is rather suggestive of the pattern found commonly in Ayrshires (Figs. 3 and 4). This resemblance was so marked that visitors on casual examination often mistook them for Ayrshires. This atypical pattern in the red and white Holsteins will be discussed later.

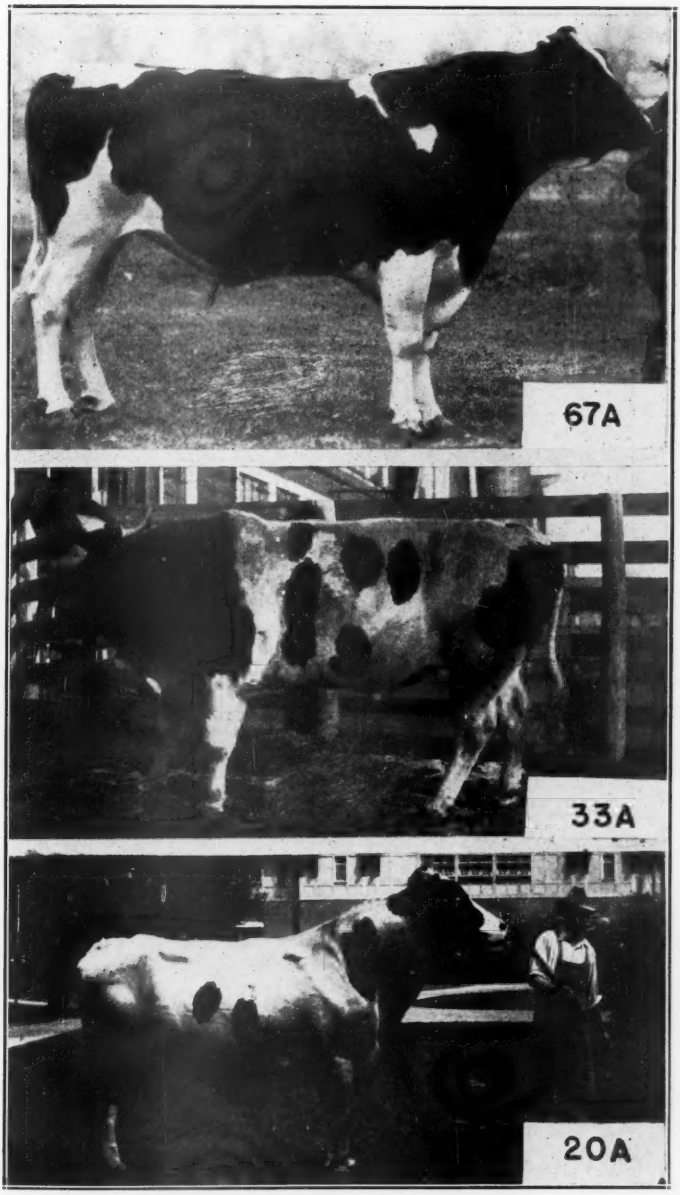

FIG. 2. Three of the black and white foundation Holstein bulls: 67*A*, 33*A* and 20*A*.

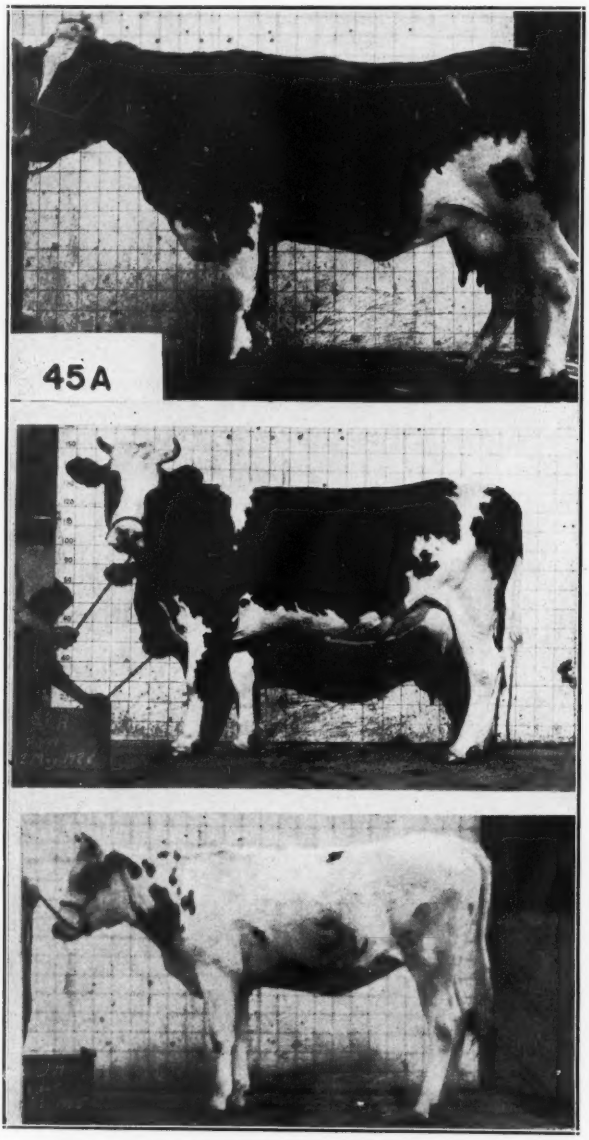

FIG. 3. Three of the red and white foundation Holstein cows: 45*A* with a high, 52*A* with a medium and 60*A* with a low level of pigmentation.

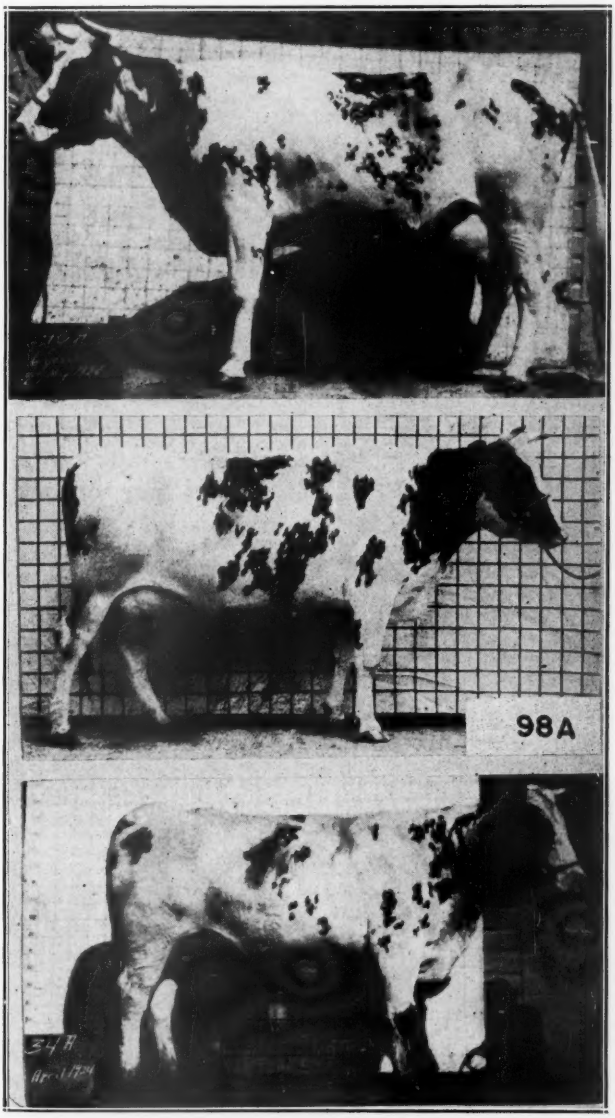

FIG. 4. Three red and white Holstein cows (40*A*, 98*A* and 34*A*) showing "Ayrshire pattern."

The F_1 generation.

All F_1 animals are the result of matings between self-colored and spotted parents, but in regard to color the F_1 generation must be divided into two groups; one group is the result of crosses between black pigmented parents and the other between black and red. Without exception the 41 F_1 animals with color description were black. Most F_1 calves showed a brownish tinge of the hair, especially on the lower parts of the body, but it is doubtful if this is of any significance since it is found also in purebred Angus calves and it was quite pronounced in the F_1 calves from the cross between black Angus cows and black-and-white Holstein bulls. The brownish color disappeared gradually during the growth period and especially at the time of the first coat shedding. Most of the F_1 animals became intense black but a few showed some brownish tinge throughout life.

Self color proved to be strongly but not completely dominant to white spotting; although most of the foundation Holsteins were predominantly white, their F_1 offspring had only a small amount of white. Figure 5 shows three F_1 cows with varying amount of white spotting. Every F_1 animal had some white in the inguinal region. Some of them had a considerable amount of white on the underline, and in a few cases the white extended out on the flanks. Seven F_1 animals had a small white spot (star) on the forehead and 13 had some white on the hind feet. Nineteen animals had black pigment on the tongue, 5 had "flesh-colored" tongue, and for the others there is no description of tongue color. There is an indication that white markings on the legs and unpigmented tongue are correlated. The muzzle was black pigmented in all cases. White and Ibsen (1935) have studied crosses between Holsteins and self-colored Galloways. They observed that several F_1 animals carried some white, and their explanation is that the Galloway parents were heterozygous for white spotting; dominant modifiers are postulated which reduce the white area in

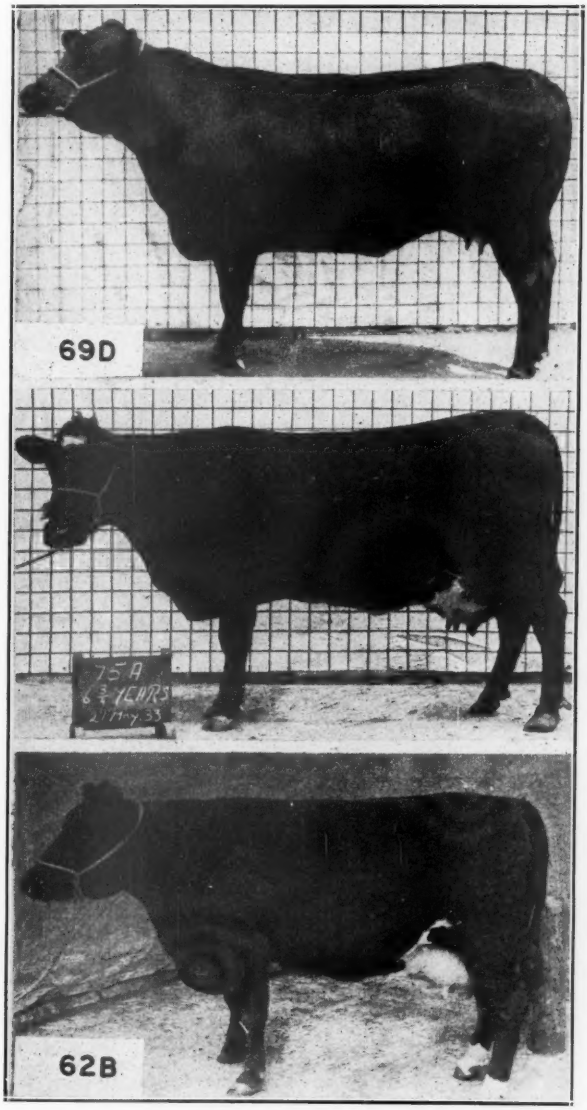

FIG. 5. Three F_1 cows from the Holstein × Angus cross: 69*D* with inguinal white, 75*A* with star on forehead and white underline and 62*B* with white udder, white underline and white hind feet.

purebred Galloways and F_1 animals. This hypothesis does not seem to fit the facts as well as simply assuming that self-color is incompletely dominant to white spotting. The frequent occurrence of "inguinal white" in the Galloway or Angus breeds may or may not have a strictly genetic explanation.

The F_2 generation.

The F_2 generation comprises 43 animals with color descriptions, but 7 of these were offspring of the F_1 cows 25*C* and 43*D*, which were both daughters of the black Angus cow 16*A* mated to the black and white Holstein bulls 20*A* and 33*A*. According to their ancestry these cows should be homozygous black, and mated to heterozygous F_1 bulls, we would expect only black offspring. Cow 43*D* mated to 48*A* ♂ fulfilled this expectation, but the other cow, 25*C*, produced in 4 matings to 48*A* ♂, 3 black-pigmented and one red-pigmented calf (59*D* ♂). Accordingly this cow and at least one of her purebred parents must have been heterozygous for black (*Ee*).

Since 25*C* was heterozygous her offspring from matings to the F_1 bull, 48*A*, may be included in the F_2 population. Subtracting, therefore, the 2 black calves of 43*D* from the total leaves 41 F_2 animals from matings between F_1 parents, which all should be heterozygous for black. These 41 fall into two distinct groups, when only the color and not the markings is considered, 24 black and 17 red. There was no intergradation of color as in the Jersey × Angus cross and therefore there can be no error involved in the classification; an F_2 animal in the Holstein × Angus cross is either non-diluted black or red. Only one of the F_2 animals showed a somewhat diluted red (112*A*). No cases of brindling appeared, but three of the 16 red F_2 animals showed some dark pigment on head and legs. These animals had also dark or mottled muzzles. Two others are recorded as having dark and one a mottled muzzle.

The ratio 24 black : 17 red is a poor fit to be expected 31 : 10 on a monohybrid basis ($P \cong 0.02$). There is,

however, no evidence from other experiments of a more complicated relationship between red and black pigmentation, and therefore we prefer to adopt the simple 3:1 ratio. Since only one calf, aborted after six months gestation, is without color description, selection can not be assumed to have influenced the results. Lloyd-Jones and Evvard (1916) reported in F_2 from the Iowa Galloway × Shorthorn cross 15 black : 6 red, and Campbell (1924) obtained in the Illinois Holstein × Guernsey cross 21 black : 8 red. These ratios each realize the Mendelian expectation as closely as the numbers allow, but when added together they show slight excess of red segregates.

The inheritance of white spotting.

Studying the inheritance of white spotting all the 43 F_2 animals can be treated as one group. At first glance there appears to be an almost continuous gradation from individuals that are self black (or red) to those that are almost wholly white. Closer inspection, however, makes possible a separation into two groups, one resembling the Angus and the F_1 in being either wholly pigmented or with relatively little white, and the other spotted with white within the range observed in the Holstein breed. The animals in this latter group in no case have less than about one-fourth of the total skin area white and there is always some white on all four feet immediately above the hoof. In purebred Holsteins with a high level of pigmentation the black may extend well below the knees, but it seldom extends much if at all below the hocks. One of our F_1 cows (62*B*) had white hind feet, but no white on the fore legs, and the F_2 steer 106*A* had both hind feet and the left fore foot white, but had very little white on the underline. We have accordingly classed both these phenotypically in the "F_1" group. The animals marked like Holsteins we call *spotted*, and for convenience we may refer to the other group as having the "*self*" pattern, though the word is used here in a relative sense only.

On the basis of the above distinction 33 of the F_2 animals may be classed as self (*SS* and *Ss*) and 10 as spotted (*ss*). This approximates closely a 3:1 ratio. We may assume therefore that we are here dealing with a single major gene difference for spotting, the Angus being *SS* and the Holstein *ss*. It is necessary to assume also a number of modifying genes which determine the grade of pigmentation, and in addition a certain amount of non-genetic variation. It is surprising, however, how true the spotted F_2 segregates are to the general pattern of Holstein-Friesian animals (Figs. 6–8). This would seem to show that neither the Holsteins nor the Angus foundation animals carried any important modifying genes for localization of the pigmented areas.

Individuals classified as spotted range from 101*A*, with the least white included in this classification, to 59*D*, with the most white, in the following order: 101*A* ♀, 92*A* ♀, 91*A* ♀, 76*B* ♀, 83*A* ♀, 70*A* ♂, 72*C* ♂, 59*C* ♀, 59*A* ♂, 59*D* ♂ (*cf.* fig. 2, Part I). Seven of these animals are shown in Figures 6–8. The cow 76*B* had about 40 per cent and the steer 91*A*, 70 per cent of the total skin area pigmented (black); 76*B* had pigmented spots on the legs. The bull calf 59*D*, which died at two days of age, was almost wholly white with a few red spots on the head and the neck only.

The "self-colored" 33 F_2 animals, comprising the dominant class, are difficult to classify according to the amount of white, as they form a graded series with no clear class distinctions. The following will, however, give some idea of the distribution of white on them:

	Self colored	Inguinal white	White on underline	White markings on legs or forehead or both	Total
Red	3	4	2	4	13
Black	1	6	6	7	20
Total	4	10	8	11	33

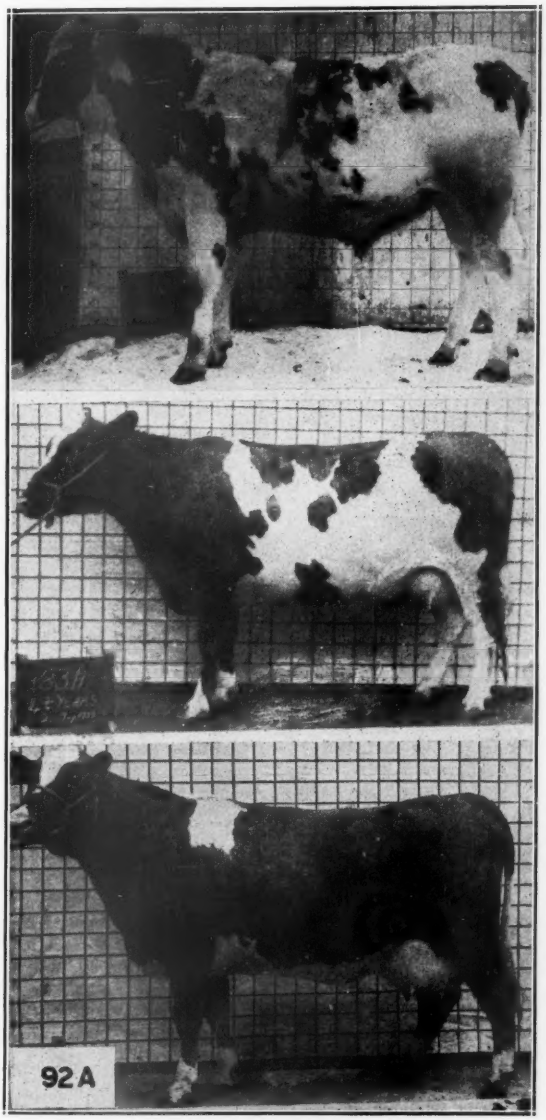

FIG. 6. Three spotted red and white segregates from the Holstein × Angus cross: the steer 72*C* (with ragged pattern), and the cows 83*A* and 92*A* with a higher level of pigmentation than 72*C*, but ragged borderline between the red and white areas.

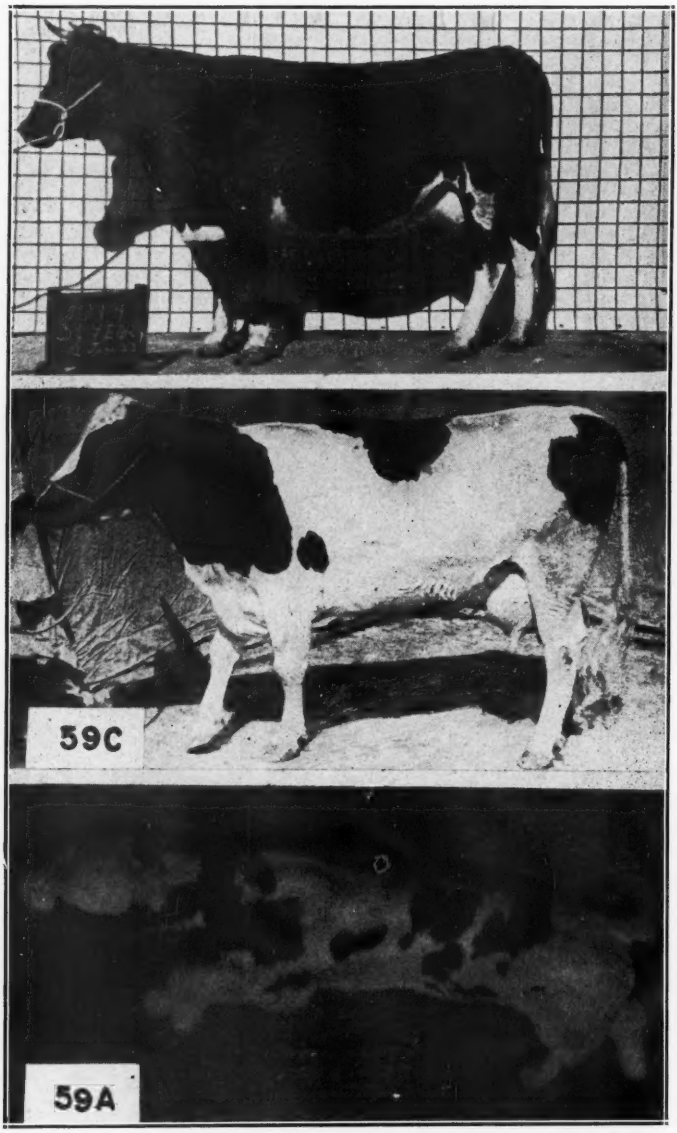

FIG. 7. Three spotted black and white F_2 segregates from the Holstein × Aagus cross: the cows 101*A* and 59*C*, and the bull calf 59*A*, which died one day after birth. The cow 101*A* shows the least amount of white among the *ss* segregates.

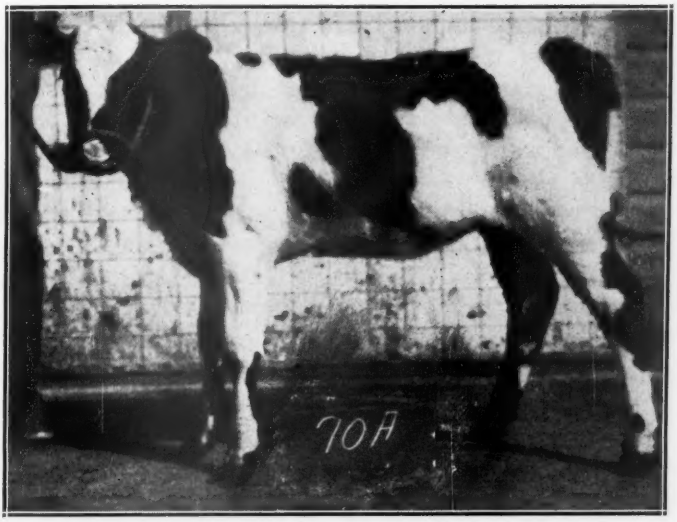

FIG. 8. Pigmented legs in the spotted black and white F_2 heifer 70*A* from the Holstein × Angus cross.

Our conception of the action of the recessive gene for white spotting (*s*) is that it releases, so to speak, a generalized invasion of white, or inhibition of pigment formation, over the body in the manner suggested by Allen (1914). The amount of white is conditioned, except for an undetermined degree of nongenetic variation, by other factors (extension modifiers). Without specific genes there is no definite pattern except that resulting from the recession of pigment towards the "pigmentation centers." This recession of pigment, or extension of white, occurs in a more or less definite order. It would appear that *S* is not completely dominant over *s*, and the earlier stages in the series are accordingly represented in our F_1 animals and in many of the "selfcolored" F_2's. Most purebred Angus may have a small amount of white on the udder of the cow or the "cod" (scrotum) of the bull, indicating a variability in the threshold for complete action of *S*, which may be due to the interaction of several genes, or perhaps of somatic origin. The invasion

of white in the F_1 animals is in approximately the following order: a few white hairs or a small white spot in the inguinal region; big splash of inguinal white; white extended to umbilicus; white on hind feet; on switch; on fore legs; white star on forehead; white extended to flank and elbows.

The further recession of the pigmented areas occurs in the *ss* segregates in F_2 and is represented in the characteristic pattern of Holstein cattle. The white extends completely along the underline, appears on the legs and begins to run into the "primary breaks" between the pigment centers. As the white extends still farther the primary patches become separated and usually begin to break up into secondary spots. There appears to be a tendency for the patches on the head and neck to persist and to remain relatively intact. According to Allen five pigment centers may be recognized in most mammals: bi-lateral aural, nuchal, scapular, pleural and sacral centers. Deakin (1944) points out that in "ideally colored Holsteins" there are three pigmented areas, *viz.* the neck, "saddle" and rump spots. This would seem to describe the situation in Holstein cattle even better than Allen's scheme. At any rate it must be recognized that the variability is quite considerable. The point we wish to make is that the type of spotting found in the "Lowland breeds," and which is characteristic of the Holstein-Friesian in America, and other direct derivatives of the Dutch-German Friesians, represents what may be called a physiological pattern due to the simple recessive invasion of white resulting from the gene *s*, with few if any modifiers except those which determine the general level of pigmentation. Other breeds, such as Guernsey and Ayrshire, appear to have the same general type of pattern but modified to some extent by factors affecting the distribution of the pigment. For convenience the generalized type may be designated the "Friesian pattern." The only alternative explanation of the same facts would be to state that the Holstein-Friesians

are homozygous for a recessive gene for the "Friesian" type of pattern, a gene which may or may not be present in other breeds with recessive white spotting, *e.g.* Guernsey and Ayrshire. We prefer, however, the explanation discussed above.

Animals having the generalized "Friesian" pattern, as we have defined it, probably do not carry specific factors for the localization of the spots. The occurrence of white on the different parts of the body is determined, within developmental limits, by the level of pigmentation. A correlation is therefore to be expected between areas of "weakness," in the general order in which pigment recedes and white extends. Thus an animal in which the white extends far up the hind legs is more likely to have considerable white on the underline and at least some on the front feet; if the front legs are white to the knees there is more likely to be a star or blaze on the forehead, a white patch on the withers, on the loin, etc. Walther (1913) pointed out this sort of correlation in horses and Lauprecht (1926), Wiesch (1929) and Sommer (1932) have similarly found correlation between the amount of white on the head, throat and legs and the total amount of white spotting in the Lowland breeds of cattle. In view of these facts it is obviously the wrong approach to attempt to analyze the inheritance of particular spots when only the general recessive type of white pattern is involved, and attempts to fit multiple factor explanations to particular sets of data, as is done by Funkquist and Boman (1923) and Funkquist (1927) in their study of white markings on the head in the Swedish Friesian and Swedish Red and White cattle, have little meaning.

From a study of the color markings of double monsters Kröning (1924) concluded that there is considererable non-genetic variation in the white pattern, but Nachtsheim (1924) points out that these double monsters really show a striking similarity, and that Kröning overemphasized the non-genetic variability. A considerable body

of unpublished material in the Department of Genetics of the University of Wisconsin would seem to support this conclusion. Kronacher and Sanders (1936) studied a great number of cattle twins and they found that within monozygous pairs there was a great similarity of pattern as well as of percentage area of white but also a considerable variation in the configuration of certain pigmented or white spots. Kronacher and Sanders concluded that while the type of pattern is determined by heredity, the finer details in the extension of pigmentation are subject to non-genetic variability. Bonnier and Hansson (1946) in their investigations of monozygous cattle twins have made the same observation.

Some attempts have been made at a genetic analysis of the level of pigmentation in cattle. Wiesch (1929) found no evidence of dominance of high level of pigmentation over a low level in a study of black and white East-Friesian cattle. He states that the assumption of three pairs of multiple factors for the amount of white very well fits the actual data. Using planimeter measurements of color sketches, Pfähler (1931) found a dam-offspring correlation of 0.34 ± 0.09 for the amount of white in the Simmentaler breed and 0.36 ± 0.14 in black and white Friesian cattle. These correlations indicate a rather strong hereditary determination of the level of pigmentation. Pfähler concludes that several genes must be involved but does not attempt a definite factorial explanation. Dunn, Webb and Schneider (1923), in a study of level of pigmentation in Holstein-Friesians, found some indication of dominance of the darker type over the lighter one. The extreme spotted type appeared to breed true, a further indication of its recessiveness. These authors favor an interpretation based on one or more major factor or factors with partial dominance and further blending due to modifying factors. Briquet and Lush (1947) in a similar study of the same breed reached the conclusion that the heritability of individual differences in the percentage of white is probably above

90 per cent, not much room being left for dominance, epistasis or non-genetic variation. They also state that: "To explain the results in forms of Mendelian factors seems to require several pairs of genes." We are inclined to the same view, and not knowing the number of modifiers for level of pigmentation we refrain from assigning gene symbols to these modifiers.

"Pigmented leg" and Inguinal white."

With a high level of pigmentation, the black in Holstein-Friesians may extend below the knee and hock, but they always have some white on the feet. In two of the F_2 segregates, the heifers 70*A* and 76*B* (Fig. 8), there has appeared a type of pigmentation of the lower legs entirely foreign to the Holstein breed. This consists of a rather characteristic, broken spotting, and may occur with a relatively low general level of pigmentation. This condition was doubtless caused by a gene brought in from the Angus, probably the same as the *Pl* gene postulated by Ibsen (1933) to explain a similar marking in Ayrshires and the Shorthorns. Ibsen states that the *Pl* gene "also causes some irregularity of the white spotting on the head by adding small pigmented areas." Such irregular borders and small spots on the head are, however, not apparent in our segregates that have the pigmented legs. In crosses between black-and-white Friesian bulls and red-and-white Breitenburger cattle, similar in pattern to the Ayrshires but with a higher level of pigmentation, the F_1 animals showed typical "pigmented legs" (Butz and Heinmüller, 1937). Apparently the gene for pigmented legs was dominant in this cross. The *Pl* gene did not cause any irregularity in outline of the white on the head or elsewhere in the F_1 animals as observed by Ibsen (1933) and by White and Ibsen (1935).

Ibsen (1933) accepts Gowen's (1918) evidence for a specific dominant gene for "inguinal white" and designates it *In*. The fact, however, that crosses between self-colored and spotted breeds produce an F_1 genera-

tion with inguinal white does not prove that the spotted breed carries a particular dominant gene for inguinal white, but would rather seem to indicate that self color is not completely dominant (*cf.* White and Ibsen, 1935). Ibsen's citation of a ratio of "twenty-one with the spot and six without," from the mating together of "heterozygous inguinal spotted animals," loses most of its force if one examines Gowen's table carefully. Ibsen himself points out that the presence of white in the underline makes it impossible to determine the inguinal spot, and the table shows that the animals mated had all degrees of white on the belly. Until more evidence is available to the contrary, we incline to the view previously suggested that a small amount of inguinal white which is a common occurrence in *SS* animals, such as the purebred Angus and Galloways, should not be attributed to a definite gene for inguinal white and that the variable amount in *Ss* crossbreds, depends on a threshold value determined by the modifiers of pigment level present, and probably also to some extent on non-genetic variability. The same explanation may account for a small star on the forehead. Bushnell (1940) assumes that some Holsteins carry a dominant gene for "white star," but only one of his F_1 animals showed some white on the forehead, and the dam of this animal was nearly white with small black spots. This would seem to be too slight evidence for a gene hypothesis. A genetic analysis of "inguinal white" should be made within a "self-colored" breed, *e.g.* the Aberdeen Angus. Our foundation Angus animals had, with a few exceptions, some white in the inguinal region.

The relation between color and type of pattern.

It has already been mentioned that the red-and-white foundation Holstein cows had a pattern atypical for the Holstein breed. This was particularly true of the five cows, 34*A*, 40*A*, 41*A*, 60*A* and 98*A* (Figs. 3–4), which all had a low level of pigmentation. Not only are the red spots in these animals small, but their borders are

very irregular, presenting a "ragged" appearance. This is especially evident in the neck, shoulders and sides. It will be recalled that in black-and-white Holsteins with a very low level of pigmentation the spots tend to break up, but their borders are relatively regular. With a high level of pigmentation in the red-and-white animals there is less opportunity for the ragged character to be expressed, but the pigmented border is much more irregular in the red-and-white animals.

One of the most striking results in the F_2 generation was that the red-and-white spotted segregates (note particularly 72*C*, Fig. 6) showed the ragged pattern while the black-and-whites had the more regular spotting.

Esskuchen (1929) has pointed out that in black-and-white breeds the borderline between the pigmented and white areas is more regular than it is in red-and-white breeds. This ragged border is particularly evident in Ayrshires, which commonly have a relatively low level of pigmentation. It is less apparent in those breeds which have a high level of pigmentation, such as the Guernsey, but if a series of Guernseys be examined it will be noted that in general they differ from Holsteins of the same level of pigmentation in having much more irregular borders to the pigmented areas. In view of these facts we are led to the conclusion that this distribution of pigment is rather a physiological accompaniment of the red color. The ragged border between red and white areas may, however, be modified to some extent by other factors, such as those determining "block" and "broken" spotting. Our hypothesis explains very simply why the foundation red-and-white animals, particularly those which had relatively little pigment, resembled Ayrshires. Incidental records of some other red-and-white Holstein calves indicate that they also had the ragged pattern. In other breeds, however, there may be other genes which modify the character of the spotting. The studies of Ryde (1921) on the Swedish Ayrshire, for example, seem to indicate that in that breed

the extreme ragged type is recessive to the more regular outline.

Gilmore, Petersen and Fitch (1942) found that when Holstein animals were crossed with Ayrshires and Guernseys the regularity of the Holstein pattern was dominant, or epistatic, in F_1 to the more irregular pattern of the red parent breed. Butz and Heinmüller (1937) made the same observation when Friesian bulls were crossed with cows of the red spotted Breitenburger and Simmentaler breeds. It should be noted, however, that the F_1 calves with regular pattern were all black pigmented. The results, therefore, agree with our hypothesis that in cattle there is a connection between the kind of pigmentation and the regularity of pattern.

Color of muzzle and tongue.

All black, and black-and-white F_2 animals had the muzzle black pigmented unless the white blaze or snip on the head extends down so as to include the muzzle. All black-and-white animals with the Holstein pattern (*s*) have the tongue unpigmented, while self-colored animals, or those with only a small inguinal spot invariably have a black tongue. In cases of somewhat more extended white spotting the tongue may be either partially or wholly unpigmented.

For the red or red-and-white animals no distinction is made in the records between unpigmented muzzle and tongue and extension of red pigment in these regions. Most of these animals were reported to have "fleshcolored" muzzle and pink tongue. Two, however, are said to have black muzzle and pink tongue and both of these animals were dark red (105*A* ♂ and 108*A* ♂). It is not known whether they carried some black pigment or whether the pigment in the muzzle was simply condensed red. The red F_2 animals which composed the crossbreeding herd in May, 1933, were examined for muzzle color, and it was found that they all had what appeared to be red pigment in the muzzle. Some of them had, in addition, dark mottling on the muzzle; in such

cases the face also showed a slight suggestion of brindling. The tongue did not carry any black pigment.

As Ibsen (1933) points out there is presumably no particular gene for muzzle or tongue color, but the color of these parts depends on the kind and intensity of pigmentation of the animal as a whole and on the level and distribution of white spotting. In other words, white on the muzzle and tongue bears the same relation to the general level of pigmentation as do white star and inguinal spot already discussed, and a correlation of white on all of these parts is to be expected. For this reason attempts to study muzzle or tongue color as separate characters, as done *e.g.* by Funkquist (1920), can scarcely be expected to lead to definite results.

Backcrosses and other crosses.

The black F_1 bull 48*A* was mated twice to the red Angus cow 32*A* and produced one red and one black calf both with some white in the inguinal and umbilical regions. The reverse backcross of the F_1 cow 25*C* to Holstein bull 33*A* produced a black-and-white heifer calf, 53*A*. This heifer by the F_1 bull 48*A* produced three calves (74*A* ♀, 74*B* ♀ and 74*C* ♀), all black with a small amount of white on the underline.

The other matings were of F_2 cows to the F_1 bulls 69*B*, 87*A* and 87*B*. These matings were not made for the purpose of genetic analysis but simply to obtain lactation records of the cows. With the red and white cow 71*A* the F_1 bull 69*B* produced three red calves (89*A*, 89*B* and 89*C*), one of which was self red while the other two had some inguinal white. With another red and white cow, 92*A*, he produced one black-and-white bull calf (116*A*). Eleven black-and-white F_2 cows (7*S* and 4*ss*) were bred to the three F_1 bulls and produced 15 black and 3 red calves, which happens to be the ratio expected if one-third of the cows were *EE* and two-thirds *Ee*.

Considering the "Friesian" pattern, five of the F_2 cows (59*C*, 70*A*, 76*B*, 92*A* and 101*A*) classified as of this type (*ss*) were mated to F_1 bulls (*Ss*); in this case an

equality of "self" and "Friesian" pattern offspring would be expected. Of the 8 calves from these matings five (84*A*, 85*B*, 115*A*, 116*A* and 125*A*) were classed as *ss* and three (85*A*, 104*A* and 124*A*) as *S*. The 8 "self" F_2 cows by the same bulls produced one "Friesian" pattern (103*B*), 1 self and 12 with varying amounts of ventral white. This ratio of 13 to 1 is fairly close to expectation if two-thirds of these cows were *Ss*. It may be affirmed, therefore, that the results from the backcrosses are in general agreement with the conclusions drawn from the straight F_1 and F_2 generations.

Most of the backcross calves were slaughtered within a few weeks after birth and the color of muzzle and tongue is recorded in only a few cases. As far as the records go they are in accord with previous conclusions.

Summary

The results of crossbreeding between self-colored black Aberdeen-Angus animals and fawn Jerseys, and also between black, or red Angus and black-and-white, or red-and-white Holstein-Friesians may be summarized as follows:

1. In the Jersey $\times$ Angus cross the black color of the Angus breed was almost completely dominant in F_1 over the fawn color of the Jerseys. The young calves showed in some cases a considerable degree of fawning but darkened with increasing age, and most F_1 animals became intense black at the age of maturity. In F_2 segregation occurred but the gradation in color was rather continuous, ranging from solid black to dark Jersey fawn. If F_2 animals with muzzle band and distinct fawning are considered as Jersey colored, and the others as black, a ratio of 13 black to 4 Jersey colored is obtained. Two out of 4 Jersey colored F_2 animals were distinctly brindled. The results are explained on the assumption that most purebred Angus are homozygous for a gene *E* causing complete extension of black pigment over the body. The Jerseys carry a different gene, *Bs*, causing only a partial extension of black. Modifying genes may

influence the level of pigmentation in animals with partial extension of black. The gene for brindling, *Br*, was apparently introduced to this cross from the Angus.

2. In the Holstein × Angus cross black-pigmented bulls and, in the majority of cases, red-pigmented cows were used: two Angus cows were, however, self black. In matings between red- and black-colored parents, black was completely dominant over red in the F_1 generation. In the F_2 clear cut segregation occurred but the ratio of 24 black to 17 red is a poor fit to the expected 3:1 ratio ($P \cong 0.02$). Considering results from crosses between black and red breeds, reported by other investigators, however, the hypothesis of monohybrid segregation is adopted.

3. In the F_1 generation of the Holstein × Angus cross self color is strongly but not completely dominant to white spotting. Many F_1 animals have, however, a considerable amount of white on the underline, and they may also have some white on the hind feet and a small white star. In the F_2 generation there is a range in the extension of pigment from self-colored to predominantly white. The range is not quite continuous, however, and the F_2 animals can therefore be divided into two fairly distinct groups, one group resembling the Angus and the F_1 generation, and the other spotted with a variation in the extension of pigment observed in the Holstein breed. The ratio of "self" colored to spotted is 33:10, which approximates closely the expected 3:1 ratio in a monohybrid segregation.

The Angus breed is homozygous self-colored (*SS*) and the Holsteins are homozygous for a recessive gene for white spotting (*s*), the level of pigmentation (the amount of white) being influenced by an unknown number of modifying genes. In the spotted animals the pigment recedes in a fairly definite manner as the level of pigmentation decreases, primary and secondary breaks occurring between pigment centers.

The presence of pigmented spots on the legs in some

of the *ss* segregates may be explained on the assumption of a gene, *Pl*, for such pigmentation, which probably was brought in through the Angus breed. No evidence is found for the presence of a particular gene for inguinal white.

In red-and-white animals the borderline between pigmented and white areas is, as a rule, much more irregular than is the case when the animals carry black pigment. This may be due to a physiological correlation between red pigment and pigment distribution.

4. Self-colored black animals have black muzzle and tongue. In spotted black-and-white animals the extension of black pigment on the tongue is correlated with the extension of black on the body. The *ss* segregates from the Holstein × Angus cross had invariably "flesh-colored" tongue. The muzzle color is black in animals carrying the gene for extension (*E*) or partial extension (*Bs*) of black when white spots do not invade this region of the head. In the red animals the muzzle is "fleshcolored" or carries red pigment. We have found no evidence for the assumption that either muzzle or tongue color is determined by particular genes.

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MIMICRY, AND ECOGENOTYPICAL VARIATION

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DR. WM. HOVANITZ, (1945) discusses the paper by Hale Carpenter and Hobby on *Limenitis bredowii* (1944) in a very interesting way, paying special attention to those authors' conclusion that the small differences in appearance of *bredowii* in the southern part of its range from those further north are associated with the presence of species of *Adelpha* in the south. Dr. Hovanitz considers that "the convergence of *Limenitis bredowii* toward *Adelpha* is related to the physical factors of the environment; such parallel ecogenotypical color variations are shown by many animals. The convergence toward *Adelpha* then would be caused by the action of similar environmental agencies on all the butterflies and mimicry need not be considered." Reference may also here be made to another paper by the same author (1941) in which he says (pp. 278–9): "Much of the work done on mimicry in butterflies should probably also be mentioned here as perhaps being in many cases only parallel ecological variation causing convergence of color pattern appearance."

Undoubtedly such convergence exists but the geographical variations of African butterflies cannot be so easily ascribed to ecological conditions; for example, the common *Danaus chrysippus* L. A random collection from an island in Lake Victoria showed all the named forms (except *transiens Suffert*) flying together. As one proceeds northwards the more uniformly brown *dorippus* Klug becomes more abundant and in the drier parts of the Sudan is markedly predominant. So far, so good. But if the species is traced westwards into the Congo forest and the West Coast we find that it becomes repre-

sented in the latter very humid area by the form *alcippus* with the hind wings largely white, and that is the only form on the West Coast.

Again, let us consider that famous African species, *Papilio dardanus* Brown, of which several geographical races are known, distinguished in the males by the different development of the black pattern on the light yellow ground. It might be expected, on Dr. Hovanitz's premises, that the West African race *d. dardanus* Brown would be more strongly marked with black than the East African *d. tibullus* Kirby which inhabits country where there is a marked dry season. This is not the case, however, for *tibullus* is the most heavily marked with black of all the races.

When mimetic resemblances are considered in detail many queer facts come to light which do not seem to harmonize with the facile explanation that has been put forward, and it is certainly not adequate by itself. Thus, the co-existence in one locality (Entebbe in Uganda) of several species of the Acraeid genus, *Bematistes,* with similarly colored forms of one species of Nymphaline, *Pseudacraea eurytus* Linnaeus. Some of the former are alike in both sexes, others are dimorphic, and there are forms of *eurytus* corresponding to the sexes of each *Bematistes.*

In the same locality several female forms of *Papilio dardanus* mimic other species of Danaididae or Acraeidae, while the males are not mimetic. Now it has been shown that at Entebbe the super-abundance of the models *Bematistes* coincides with a high degree of perfection of mimicry in the *Pseudacraea,* but that on the Sese Isles of Lake Victoria, only some 20–30 miles south of Entebbe, scarcity of *Bematistes* is associated with diminution of the mimetic likeness in the *Pseudacraea* and great variability (Poulton, 1912; Carpenter, 1914). Similarly, the females of *P. dardanus* in the highlands of Kenya are remarkably variable in the absence of the species usually serving as models (Ford, 1936). A case analogous to one

that was noticed long ago in South Africa by Roland Trimen (1887), the first to observe mimicry in Africa, may aptly be quoted here. The males of two Papilios are with difficulty distinguished from each other but the female of one, *P. zoroastres homeyeri* Plötz mimics a Danaid, while the female of *P. cynorta* Fab. mimics an Acraeid. Both occur in the same localities. A somewhat more complicated state of affairs is exemplified by *P. cynorta* and its model *Bematistes epaea* Cramer in different parts of Africa. On the West Coast *B. epaea* has reddish brown male and black female, with simple white pattern. The female is closely resembled by the same sex of *P. cynorta* of which the male is quite different and non-mimetic. These species if traced eastwards into Uganda undergo a change: the *Bematistes* becomes grey-brown with the areas that were white in the West now much contracted and pale yellow, both sexes are now alike: it is the race *paragea* Grose Smith. In the case of the *Papilio* there is the same change in the female, but the male is almost indistinguishable from the West African male. These same species occur in Abyssinia. The *Bematistes* is monomorphic here also, but instead of both sexes being altered to a common facies the male preserves the red brown of West Africa and the female has adopted the same color. But the Papilio goes back to black and white, and does not resemble the black and white species to which it is similar in West Africa (*B. epaea*) but agrees in pattern with the Abyssinian form of the black and white Danaid *Amauris n. niavius* Linnaeus. The peculiar circumstance in all this is that *Amauris niavius* is abundant on the West Coast (where it is the model for *Papilio dardanus*) but that in that area *Papilio cynorta* does not mimic it, but *B. epaea!*

There is yet another complexity in this association discussed by K. Jordan (1911). While the West African *B. epaea* has two forms with different coloration, these forms are sex-limited, the male being red brown, the female black and white. There is also in the same locality

a species of Elymniinae—a group common in Asia but represented in Africa by only one species, *Elymniopsis phegea* Fabricius. This closely resembles, in coloration and pattern, both sexes of *B. epaea,* but a red brown specimen may be of either sex, as may the black and white individuals. *E. phegea* is also found in Uganda, together with *B. epaea,* but as Jordan says, there is hardly any resemblance between the Uganda *Elymniopsis* and *B. epaea paragea.* Jordan explains the discrepancy by pointing out that in Uganda there are several other species of *Bematistes,* commoner than *paragea,* which serve as models for the *Elymniopsis.*

Before passing on to some general considerations exception is taken to a statement by Hovanitz in 1940 (pp. 376–7): "The actual proof of selection by predators (or by any other means) on any living thing in nature has not yet been shown; in fact, in most cases of supposed protection by protective resemblance, it has never been shown that the actual effective predator is selective on account of the supposed protection." One wonders whether Dr. Hovanitz is familiar with the experiments conducted by Carrick on cryptic caterpillars on a branch placed at the approach to the nest of a wren which ignored them in their resting position but took them when pinned on to a board so that they were freely visible? (Carrick, 1936).

The following account is quoted from a paper on experiments on the edibility of insects to a young monkey in Africa. (Carpenter, 1921). A young monkey, quite unafraid of the writer with whom he was familiar, was taken out 'hunting' in the bush, at the end of a long string which gave him freedom to go exactly where he pleased followed by the writer who noted what happened. January 4, 1917. "At 4:30 p.m. I took M. out hunting. He had had plenty of vegetable food in the afternoon, but no insects. Obs. 156 sp. 6. Meloid: on the first bush were several of the beetle *Coryna dorsalis* Gerst. I induced M. to put out a hand and touch one, but he would not even smell it. Obs. 157, sp. 42. Coreid, *Anoplocnemus*

curvipes F.: M. saw this bug, caught it, and ate it very quickly, as if afraid of being pricked. Obs. 158. He chased and ate a small Acridian, then at some grass. Obs. 159, sp. 78. An Arctiid caterpillar, about an inch long, was touched and left. Obs. 160, sp. 16, Acridiid, *Phymateus viridipes* Stal: a pair of these aposematic grasshoppers were *in copula* freely exposed on short grass in the open. M. went up to them and pawed the male. Without attempting to get away the grasshopper merely erected its wings perpendicularly so as to display their purplish and black colours. M. took no more notice and ate some grass. Afterwards he ate other insects, including a large *Cyrtacanthacris* grasshopper.''

This account, admittedly, concerns insects whose protective coloring is not crytic but aposematic. But the following observation (p. 46) is apt. ''Jan. 29, at 2 p.m., M. having had a good meal of vegetable food but no insects. Obs. 533, sp. 59, Lagriid, *Lagria rhodesiana* Péring, a dull purplish, hairy, soft beetle that freely exposes itself on grass tips. I kept this in my closed hand, so that M. was inquisitive to see what was inside. When I opened my fist and he saw the very distasteful beetle his face fell and he would not touch it. But he took it as a joke, broke into a broad grin, and frolicked with my hand in a most amusing way. Obs. 539, sp. 260. Buprestid, *Discorderes* sp., not in Br. Mus.: a highly procryptic beetle of rather curious appearance, about half an inch long, brown, with curious little knobs and excrescences. I found a pair *in copula* on a leaf, and did not recognize them at first; nor apparently did M. I offered one on my hand; M. looked again at it, and put it in his mouth for a preliminary bite. This, however, made no impression on the hard beetle, and M. took it out for another look. After several more gentle bites had no effect M. bit harder, and finding it tasty, ate it with relish.'' It seems extremely likely that M. would have altogether passed by the beetle in its natural surroundings, owing to its procrypsis.

As to general consideration, it is quite clear that there is much to be said for Dr. Hovanitz's views on mimicry and that local conditions will certainly play some part in the production of the general appearance of the butterflies of any area. But that is far from accounting for *details* of mimetic resemblance, and particularly for the production of the same effect by markings on the mimic in a different anatomical relation to the appropriate markings in the model. (*e.g.* Dixey, 1920; Poulton, 1931). How much more does this apply to cases of mimicry outside Lepidoptera altogether, such as the two Australian Longicorn beetles with the typical banded coloration of their wasp model in one case across the exposed abdomen in the other across the closed elytra? (Fisher, 1930). The production of ant- or wasp-mimicry by different devices of structure, coloration, *and behaviour* is quite beyond Dr. Hovanitz's hypothesis. It is perfectly true that in the first days of enthusiasm over the new discovery any kind of resemblance between insects was hailed as "protective mimicry," often without a suggestion that the supposed "model" possessed any character that would make it worth imitating. Students of mimicry have, I hope, got beyond that stage now.

Finally, is it not well to remember that natural selection will, *ex hypothesi,* act upon *any* heritable variation that confers advantage? Thus, a general darkening may be brought about by environmental conditions, but cannot Natural Selection so guide the arrangement of the darker pigment in the unprotected species that mimicry of the protected species results? Does not the true solution, as regards these two points of view, lie in the old quotation, *"in medio tutissimus ibis"?*

There may be added to these remarks a new conception of the meaning of similarity of coloration put forward by the writer (1947).

Cases of general similarity in appearance exist in which it is scarcely justifiable to talk of mimicry: for example, the innumerable black and yellow Syrphid flies,

many of which can hardly be mistaken for a wasp. It is suggested that the explanation lies in 'safety in numbers': a predator can eat a certain number only of individuals with any particular coloration, and the rest will escape. The term 'ochlosis' (from Greek 'ochlos', a crowd) has been devised for this: a species is 'ochlotic' when adapted for living in a crowd, or 'ochlo-chromatic' in coloration. This principle, if valid, considerably relieves the strain on the theory of mimicry by lopping off the weaker branches; it is very possibly applicable to *Adelpha* and *L. bredowii*.

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